MAXIMUM CONSTITUENT LEVELS
AND THE LOW-YIELD CIGARETTE

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I. THE SCIENTIFIC LITERATURE DOES NOT PROVIDE PERSUASIVE SUPPORT FOR THE IMPOSITION OF MAXIMUM CONSTITUENT LEVELS

Tobacco smoke is a highly complex mixture containing thousands of constituents. Although tobacco smoke constituents are among the most heavily researched substances in the world, no constituent as found in cigarette smoke (including "tar") has been scientifically proven to cause cancer or any other human disease. Nor has any "health benefit" from switching to low "tar" cigarettes been scientifically established.[1] Thus, legislation establishing maximum levels for smoke constituents is insupportable on public health grounds.

Anti-smoking advocates frequently assert that nicotine or carbon monoxide (CO) cause disease in humans. The same is also said about "tar," even though it is a laboratory product and not an actual constituent of tobacco smoke. Over the years, much time

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1. Philip Morris does not represent low "tar" and nicotine cigarettes as being "safer." Low "tar" and nicotine cigarettes are on the market in response to consumer demand, which has increased in recent years. Because of these changes in consumer preferences, Philip Morris has modified its production and shifted its emphasis in advertising. The current popularity of some of Philip Morris' newer brands is consistent with trends in consumer preference for many products. Consumer tastes are changing, as demonstrated by the growing demand for "lighter" products such as low-calorie beers, sugar-free soft drinks, low fat foods and the like. In addition, much media and public attention has been given to the views of some scientists concerning these newer cigarettes. As a result, it is entirely possible that some smokers have been influenced by this publicity.

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and effort has been devoted to determining the identity of tobacco smoke constituents. This has proven difficult, however, and much remains to be learned about the structure and makeup of tobacco smoke and its effects, if any, on human health. Nonetheless, proposals have frequently been made to establish maximum levels of such substances in cigarette smoke. Such efforts are not supported by the available scientific data.

The significance, if any, of smoke constituents to human health is not yet understood. In addition, the smoking machines used to measure "tar," carbon monoxide and nicotine levels have little relationship to what humans experience when they smoke. As a consequence, "no ingredient (constituent) or group of ingredients (constituents) as found in tobacco smoke have been established as disease producing in smokers." (1) Nor has it been proven that a reduction in the levels of those substances will decrease any claimed harmful effects of cigarette smoke.

A. Nicotine

Nicotine is a natural element of tobacco and, thus, is present in tobacco smoke. Nicotine has been described as having no known chronic or cumulative effects on human health. (2) The data on the nature of nicotine's relationship to human health is inconclusive at best. Exposure of smokers to nicotine cannot be

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measured with precision, so that it is impossible to establish a scientific relationship between nicotine and any other (precise or imprecise) measurement. In fact, no correlation between the nicotine level of a cigarette or the number of cigarettes smoked and the smoker's actual nicotine intake has been established because of individual variations in puff rates, depth of inhalation, and body metabolism. (3)

Nonetheless, anti-smoking advocates blame nicotine for the development of heart disease. Yet no mechanism by which nicotine, or any other agent, is involved in heart disease has been demonstrated. Serious questions about what role, if any, nicotine plays have been raised as a result of autopsy findings of fatty deposits and other changes in the arteries of individuals who either have not smoked or could have smoked only briefly, such as infants, children, and young men killed in battle. (4) Even the 1983 U.S. Surgeon General's Report, which focused on cardiovascular disease, concedes that "the evidence for and against a primary role for nicotine in the development or acceleration of atherosclerosis is not conclusive." (5) That opinion was shared by a German researcher who has conducted research on the relationship between nicotine and the clogging of arteries. After reviewing the literature, he and his co-author concluded that "there is no established evidence which supports the hypothesis that

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nicotine has any influence on the development" of those changes. [emphasis added] (6)

The role of nicotine in the development of heart disease posited by anti-smoking advocates is further undermined by two epidemiological (population) studies. In a study that dealt with myocardial infarction (heart attack), the authors reported finding -- contrary to what they expected -- that the nicotine and carbon monoxide levels of the cigarettes smoked by their subjects were not related to the risk of heart attack. (7)

The second study, chaired by a well-known British scientist opposed to cigarette smoking, examined the serum (blood) levels of cotinine, a nicotine metabolite, in male nonsmokers and smokers of cigarettes only, cigars only, and pipes only. The study determined that the mean cotinine level for pipe smokers was significantly higher than the levels for cigarette and cigar smokers. Since studies of pipe smokers generally have not reported an increased risk of coronary heart disease, the researchers concluded that "nicotine is unlikely to be the major cause of the excess coronary heart disease mortality in cigarette smokers." (8) After re-evaluating their methodology in response to anti-smoking criticism of their study, the researchers again concluded, "we can be reasonably confident that exposure to high systemic concentrations of nicotine is not a cause of the disease." Although

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the researchers argued that their data "cannot completely exonerate" nicotine, they added that the data do "substantially reduce the weight of evidence suggesting that nicotine is a cause of coronary heart disease." (9)

Animal studies which purport to establish a causal role for nicotine in heart disease have been soundly criticized for their unrealistic and excessive test conditions. An American researcher who conducted animal studies on this subject has noted:

> "There have been some studies that have exhibited minor or questionable changes with the use of 600 or more cigarettes a day in man. This is such a large number that I think man would find it difficult to find the time to smoke them." (10)

In contrast, animal studies using realistic doses of nicotine have "failed to initiate, exacerbate, or otherwise influence" the process leading to the clogging of arteries in test animals. (11) In one such study, which was funded by the United States government, male beagle dogs fed a special diet to induce this process were exposed for two years to cigarette smoke containing low or high levels of nicotine and, in some cases, enriched with CO. According to the final report of the research laboratory which conducted the study, "the results of this study lent no support to the suggestion that cigarette smoking increases the rate of development" of this process. (12)

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The foregoing authorities demonstrate the validity of one researcher's summary: "While many studies have been done in this field, none have established nicotine as contributing to the causation, aggravation or precipitation of any cardiovascular disease." [emphasis added] (13)

B. "Tar"

"Tar" is not actually present in tobacco smoke, although the many inaccurate references to "tar" in smoke may have created that impression. "Tar" is actually a laboratory product obtained by collecting the particulate matter in tobacco smoke, either by passing cigarette smoke through a cold trap at extremely low temperatures or by using filters and a drying process. However, material collected in this way does not duplicate what humans are exposed to when they smoke:

> "[T]here is, at present, no available instrumentation permitting the separation and individual collection of the particulate and gas phases which duplicates the precise physicochemical conditions prevailing in cigarette smoke as it is inhaled." (14)

Despite its lack of relevance to smoking, laboratory-produced "tar" has been used in animal experiments designed to investigate the possible relationship between tobacco smoke and

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cancer. In those experiments, "tar" was repeatedly painted on the shaved backs or ears of test animals over prolonged periods of time. These so-called skin-painting experiments have prompted a great deal of interest, as the tumors which resulted have been said to be evidence that tumors might develop in human lungs from inhaling cigarette smoke.

The results of such animal experiments cannot and should not be extrapolated to the human situation. Even if the "tar" collected by the methods described above were present in cigarette smoke, the "tar" used in skin painting experiments is very different by the time it is studied in the laboratory. That is because after "tar" is collected, it continues to undergo chemical changes as long as it is stored. (15) The chemical and physical changes brought about in collecting "tar" and applying it to animals may well produce biological results quite different from any that may occur during inhalation. Furthermore, as an experimental toxicologist explained at the 1976 hearings, even if an effect such as tumor production is observed in a particular species, that does not necessarily mean that it might "occur either quantitatively or qualitatively in man." (16)

Skin painting experiments suffer from a number of additional weaknesses. For example, the concentrations of "tar" used in such experiments are extremely high. One researcher has

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estimated the amounts utilized to be equivalent to an individual smoking over 100,000 cigarettes per day (17). Furthermore, the skin and ears of laboratory animals are not similar to human lung tissue. Animal skin lacks the intricate clearance mechanisms of the lungs, such as the mucus blanket which coats the lining of the major airways of the lung. Even the summary report of a study sponsored by the United States government utilizing skin painting techniques conceded that there is an "uncertain relationship between tumors resulting from mouse skin painted with condensate and human lung cancer." (18) Consequently, such experiments involve applying "the wrong material, in the wrong form, in the wrong concentration, to the wrong tissue of the wrong animal." (19)

It is misleading, therefore, to draw definitive conclusions about "tar" and human disease from skin painting studies. As a knowledgeable observer of research in this area has stated: "Bronchogenic carcinoma [lung cancer] has never been produced by tobacco or its products in any experimental animal despite the multiplicity of attempts." [emphasis added] (20) All in all, the current state of scientific evidence concerning "tar" may be concisely summarized:

> "Human beings do not smoke 'tar' and laboratory reports on 'tar' yields have not been established as significant to human health." (21)

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C. Carbon Monoxide

Carbon monoxide (CO) is a tasteless, odorless, colorless gas produced by many natural and man-made sources, including automobile exhaust and industrial emissions. Burning cigarettes also produce carbon monoxide, but that amount has been described as "insignificant" compared to most other sources. (22) It is also naturally produced during body metabolism. Nonetheless, CO has received considerable attention in the scientific literature, usually in regard to cardiovascular disease (CVD). In a review of such literature, however, two public health specialists concluded that "despite the large amount of literature available, the conclusions that can be drawn as to the role of CO in human CVD remain tentative and open to varying interpretations." (23) This conclusion is supported by a similar statement in the 1983 U.S. Surgeon General's Report, which focused on heart disease:

> "Carbon monoxide is another major component of cigarette smoke for which there are some data supporting a possible atherogenic role; however, a review of recent literature on the role of carbon monoxide in arterial injury and atherogenesis leads to no consensus." (24)

The conclusion of another group which also reviewed the literature was more concise. The chairman of the American Heart Association Task Force on Environment and the Cardiovascular System reported

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that his group had concluded that the question of whether CO causes heart disease "remains unanswered even at the basic science level." [emphasis added] (25)

To support their claims about the adverse health effects of CO, anti-smoking advocates have emphasized the results of certain animal studies, particularly those of a Danish group who exposed rabbits on a high cholesterol diet to large quantities of CO. The exposed rabbits reportedly developed vascular changes similar to early clogging of the arteries in humans. (26) However, when they attempted to replicate or duplicate those changes in another series of experiments utilizing the same levels of CO but non-cholesterol-fed rabbits, the researchers were unsuccessful in doing so.

Specialists who reviewed these and other studies have concluded that "there is no evidence" to support the suggestion that exposure to low or moderate levels of CO increases the rate of development of atherosclerotic disease in man. Indeed, they contend that "sufficient evidence is available" to support the conclusion that "CO is not of pathogenetic consequence in atherosclerotic disease." [emphasis added] (27)

The foregoing authorities demonstrate that "tobacco science" does not support constituent level restrictions. One

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researcher who has tried to establish a causal link between smoking and disease concluded:

> "We assume that it is the tar which causes lung cancer, but we do not know this for certain.
>
> Until now, we have implicated nicotine in the development of cardiovascular diseases, but we cannot prove this.
>
> The same is true for CO." (28)

Thus, proposals to mandate maximum constituent levels lack a reliable scientific basis.

II. LOW-YIELD CIGARETTE PROPOSALS MISLEAD CONSUMERS BY SUGGESTING THAT CERTAIN TYPES OF CIGARETTES ARE "SAFER" THAN OTHERS

Despite the lack of reliable scientific proof regarding the alleged health effects of "tar," nicotine and carbon monoxide, proposals have frequently been made to limit the levels of one or more of these substances. In addition, numerous governments, anti-smokers and anti-smoking groups and health associations have publicly encouraged smokers to switch to the so-called low-yield cigarettes by advocating constituent labeling, league tables, "tar-reduction" programs and maximum constituent levels. As a result, some smokers have been led to believe that smoking low "tar" cigarettes is preferable to smoking higher "tar" cigarettes. These proposals, however, are without scientific foundation. A British

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toxicologist has noted that scientific data to support such proposals are not available:

> "The toxicologist who seeks an objective basis upon which to predict increased 'safety' or reduced 'hazard' for tobacco products lacks the necessary quantitative experimental or clinical evidence to enable him to approach this task on the basis of the toxicology of any specific constituents of tobacco or tobacco smoke." (29)

Governmental sanction of the low-yield cigarette suggests to consumers that a reliable scientific basis exists for switching to it. This is not true.

A. Studies on Low-Yield Cigarettes and Disease Risk

The widespread scientific disagreement regarding the significance of the available data on the low "tar" cigarette is reflected by the published opinions on this subject. These differences of opinion are highlighted in the 1981 U.S. Surgeon General's Report which focused on "the changing cigarette" and "the relative health effects of cigarettes with varying levels of 'tar' and nicotine." (30) In the report's preface, for example, a senior government official noted that over the years U.S. public health officials had gradually assumed a "more cautious" attitude toward the low-yield cigarette; the 1981 Report, he noted, "suggests an even more cautious approach to the issue." (31) These concerns are reflected in the report's conclusions and in the accompanying commentary by U.S. health officials. In her introductory remarks

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to the report, for example, the head of the U.S. Department of Health and Human Services (DHHS) stated:

> "In preparing this report, the scientists and scientific agencies of this Department have reviewed all current scientific evidence and have concluded that the search for less hazardous cigarettes has not yielded a product which can be considered 'safe.'" (32)

Subsequent reports of the Surgeon General have reiterated those conclusions. The 1983 report on cardiovascular disease noted that "epidemiologic evidence concerning reduced tar and nicotine or filter cigarettes and their effect on CHD rates is conflicting. No scientific evidence is available concerning the impact on CHD death rates of cigarettes with very low levels of tar and nicotine". (33) The 1984 report which examines the relationship between chronic obstructive lung disease (COLD) and cigarette smoking also noted that "evidence is unavailable on the relative risks of developing COLD consequent to smoking cigarettes with the very low tar and nicotine yields of current and recently marketed brands." (34) Most recently, the 1989 U.S. Surgeon General's Report concluded that "there is no clear evidence of substantial health benefits to consumers who switch to lower tar and nicotine cigarettes" as "there is no known safe level of tobacco product consumption." (35)

Other scientific groups who have reviewed the available data on the low-yield cigarette have also commented on its

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inconclusive nature. For example, a committee convened by an operating agency of the U.S. National Academy of Sciences to analyze the data on "reduced tar and nicotine cigarettes" concluded that "the evidence for switching to lower T/N cigarettes is doubtful." [emphasis added] The committee report explained that "while some large-scale studies have suggested small gains in health due to using lower T/N (or filter rather than nonfilter) cigarettes, other population-wide studies do not support this view." (36) Similarly, participants in an international workshop held to consider the advisability of a governmental "lower-tar policy" concluded with the following cautionary statement even as they encouraged the move toward lower "tar" yields:

> "The public needs to be made aware of the uncertainties of the policy, particularly those arising from compensatory smoking, and also that the benefits of smoking lower-yield cigarettes can only be small compared with those of avoiding the smoking habit altogether." (37)

Furthermore, as already suggested, there is strong disagreement regarding the nature of the relationship between the low-yield cigarette and disease mortality. This disagreement stems from data collected from epidemiological (population) studies, clinical studies, autopsy reports and laboratory analyses. As to the population studies, a scientific committee studying "reduced tar and nicotine cigarettes" concluded, based on its analysis of

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available data on U.S. respiratory system cancer (RSC) mortality trends, that "it is not possible to credit lower T/N cigarettes up to 1975 with significantly improving" male RSC mortality. (38)

Similar observations were made by a well-known U.S. anti-smoking researcher and his co-authors and a working group charged by the International Agency for Research on Cancer (IARC) to evaluate the carcinogenic risk of chemicals to humans. In their report, the U.S. researchers noted the "conflicting reports in the literature regarding the effect of the introduction of the filter cigarette, with the concomitant lowering of smoke yields, on the risk of lung cancer." (39)

Likewise, the IARC working group concluded "at present, only about 20 years after . . . substantial modifications of cigarette manufacture were introduced in some countries," that "no direct comparison of the health effects of lifelong use of modified and unmodified cigarettes is possible." (40) Although they viewed the results of several studies as suggestive, they concluded that "the health benefits from the cessation of smoking, however, greatly exceed those to be expected from changes in cigarette composition." (41)

A similar view was expressed by a group following the mortality experience of the participants in one of the largest on-

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going epidemiological studies in the United States. Although they reported that the total death rates and death rates from CHD and lung cancer of the participants were "somewhat lower" for smokers of low "tar" cigarettes than "high" tar cigarettes, they concluded, "It is quite apparent that reduction in the tar and nicotine of cigarette smoke did not make cigarette smoking 'safe' for the men and women in this analysis, all of whom were over the age of 40 in 1959." (42)

More recent studies examining the relationship between the low-yield cigarettes and disease rates continue to undermine the validity of the position of maximum constituent level advocates regarding the relative merits of low-yield cigarettes. For example, a review of four cross-sectional and five prospective studies regarding the evidence on the relationship between smoking low-yield cigarettes and COLD concluded that there was "none for an effect on mortality" from that disease. (43)

A 1989 report on a study on the relationship between low-yield cigarettes and nonfatal myocardial infarction (heart attacks) in women also noted this lack of data:

> "To date, there is little epidemiologic evidence available concerning the effect of smoking low-yield cigarettes on the risk of coronary heart disease."

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Nor did the data from the study provide support for those who advocate maximum constituent levels; the women in the study who smoked low-yield cigarettes reportedly did not have a lower risk of a first nonfatal myocardial infarction than women who smoked higher-yield cigarettes. The directors of the study, who are opposed to smoking, concluded that it "offers substantial evidence against" the claim of lower risk in smokers of low-yield cigarettes. (44)

Autopsy studies have also prompted criticism of the "conventional wisdom" about the health effects of the low-yield cigarette. In a well-publicized study published in 1979, an American pathologist and his co-authors compared specimens of bronchial epithelium obtained during the autopsies of about 150 regular cigarette smokers who died during the period 1955 to 1960 to those from about 180 smokers who died during 1970-1977. (45) In their study, they reported finding changes which they regarded as precancerous far more frequently in the smokers who died in the Fifties than those in the Seventies. They contended that this reduction in the latter group could be attributed to the decline in "tar" and nicotine levels over that same period of time. The article does not explain, however, why invasive or very early bronchial carcinoma was actually found more frequently in sections taken from low "tar"/nicotine smokers than from high "tar"/nicotine smokers, although the number involved was very small. (46) Nor did the re-

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searchers explain why their overall conclusions are inconsistent with observed trends in lung cancer mortality rates. Lung cancer mortality rates should have fallen sharply in recent years, if their theory is correct. However, this is clearly not the case. The significance of these findings has been challenged for other reasons as well. For example, it has been reported that these same changes occur in nonsmokers, (47) and one of the study's co-authors has conceded that they have been unable to show that these changes actually progress to lung cancer. (48)

Experimental data also seem inconsistent with the concept of the low-yield cigarette as being "safer." A Finnish occupational health specialist who recently reviewed the results of relevant laboratory studies was unconvinced by the data. In her review, she analyzed data from mutagenicity tests which some contend provide a reliable short-term method of testing for substances capable of transforming human cells to malignant or cancerous ones. After analyzing the data, she observed that "the experimental evidence on the mutagenic activity of cigarette smoke condensates produced from low-, medium- or high-tar cigarettes clearly shows, . . . , that the activity measured on a weight-for-weight basis is similar or possibly even higher in some very low-tar CSCs." Based on those results and the results of certain other tests, she concluded that "the issue of smoking-induced cancer in relation to reductions of tar in cigarettes is still controversial." (49)

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B. Machine Measurements and Human Smoking Behavior

Technical and scientific uncertainties abound regarding the low-yield cigarette. These begin with the popular but scientifically incorrect notion that the exposure of smokers to various cigarette smoke constituents has been determined by laboratory analysis. However, as researchers have pointed out, laboratory procedures cannot faithfully duplicate smoking as it actually takes place outside of the laboratory. There are simply too many uncontrolled factors which vary considerably among smokers and even in individual smokers, including the number of cigarettes smoked per day, the frequency of puffs, the depth and degree of inhalation and the length that each cigarette is smoked (i.e., the butt length). (50) In contrast, "tar," nicotine, and CO levels appearing on packets and in advertisements are obtained by smoking cigarettes mechanically under a set of defined laboratory conditions.

The machines used for these laboratory measurements are designed to compare cigarette brands in their smoke yields. Such comparisons are possible only if the factors other than brand differences noted above are controlled. However, under such conditions required for the particular purpose of ranking cigarette brands, it is not possible to measure accurately the delivery of smoke to

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any actual smoker. This situation was succinctly summarized in a recent publication on this subject:

> "In the United States, cigarette yields are determined by the Federal Trade Commission (FTC) according to standard procedures similar to methods used in other countries. The results of such tests do not represent the full behavioral range of individual smokers; as the FTC itself recognized, they simply indicate the relative yield position of brands according to a convention of analytical standards, but not actual conditions of smoke puffing or intake." (51)

A similar statement appeared in the 1981 U.S. Surgeon General's Report:

> "The methods used in testing cigarettes by machine may not correspond to the way persons actually smoke. There is evidence to suggest that cigarette yields measured by machine are very different from the yields that the consumer actually obtains by smoking the cigarette, due in part to the difference in patterns of smoking between testing machines and individual smokers." (52)

A recent study designed to compare the actual "tar," nicotine and CO intake of nearly 900 smokers and the FTC machine measurements of those substances illustrates the inaccuracies inherent in these measurements. The researchers reported that the FTC measurements were "poor predictors of relative intake of nicotine, CO, or tar," and observed that "the considerable variation of individual smoking behavior suggests that precise numerical

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rankings of cigarettes are not justified." (53) They concluded that their findings "reinforce previous reports and suggest that FTC measures have dubious meaning to consumers, except perhaps [for] a small fraction of smokers (2-3%) using very low yield cigarettes." (54)

The 1981 U.S. Surgeon General's Report addressed such difficulties in a series of research recommendations it made on this subject. As the Report notes, since smokers may smoke different cigarettes in different ways, "it is important to know how smokers consume each type of commercial cigarette" in order "to conduct meaningful assays of cigarette yields and of the biological activity of cigarette smoke." The Report asserts that "only when this information is available can smoking-machines be designed to yield the most accurate estimate of human dose." (55)

C. Disagreement on Cigarette Modification

With so many fundamental research issues unresolved, it is not surprising that there are even differences of opinion regarding the basic design of a low-yield cigarette. Although some researchers favor reduced levels of "tar," nicotine, and carbon monoxide, others advocate other approaches. A well-known British researcher contends that "a low-tar medium-nicotine approach has enormous potential." (56) His views are generally compatible

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with those of a group of British researchers who contend their data are "consistent" with "the proposition that the middle tar smoking population at large might be exposed to lower concentrations of tar and carbon monoxide by smoking a product such as the one used in these experiments with a relatively high nicotine yield and low tar yield." (57) A group of American scientists, however, is "very much opposed" to that approach, contending it is "counter-productive." They favor the development of a low-"tar," low-nicotine cigarette. (58) The approach favored by the U.K. group was also criticized by a well-known U.S. anti-smoking researcher at a conference called to discuss the question of "A Safe Cigarette?". Although he stated that such an approach is "a good idea with respect to tumorigenic activity," he contends "it is not known whether it is beneficial, or even benign, with respect to the cardiovascular system. " (59) Another participant at the same conference, apparently concerned about the same question, advocated research into the development of "a medium-nicotine, low-tar, low-carbon monoxide (CO) cigarette." (60)

A U.S. government sponsored and directed research program charged with the "development of a less hazardous cigarette" attempted to resolve these and other questions. The group which oversaw the program and which became known as the Tobacco Working Group (TWG) began meeting in 1968. It was composed of both tobacco industry and government specialists in a broad range of technical

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areas related to cigarette production, consumption, and testing. Some years later, the TWG was incorporated into the National Cancer Institute's (NCI) Smoking and Health Program (S&HP), where it assumed a broad advisory role. During that period, the research program investigated many different cigarette variables, including modifications in cigarette design and smoke composition and changes in the processing of tobacco and the manufacturing of cigarettes. The tobacco companies continued to provide technical expertise to the program during its existence. The program in its entirety was eventually abandoned in 1979 after the expenditure of approximately $32 million (U.S). (61)

Although the program's published reports suggest that some of the experimental cigarettes fared somewhat better in certain bioassay tests, while others did better in other types of studies, the NCI never concluded that any one cigarette design was "safer." (62) All of the work, however, was subsequently reviewed in the 1981 U.S. Surgeon General's Report. Based partially on this extensive research, the report concluded that "the search for less hazardous cigarettes has not yielded a product which can be considered 'safe.'" (63)

Nonetheless, in 1978, the director of this program and a co-worker published an article in which they argued that the "toxic" substances of a large number of cigarette brands had been reduced

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to such an extent that an individual could smoke a certain number without detectably increasing his risk of disease compared to that of a nonsmoker. (64) However, the accompanying editorial contended that the authors "made some assumptions with which many readers will not agree," including that "the harm done by cigarettes correlates directly with their content or production of tar, nitrogen oxides, hydrogen cyanide, acrolein, and carbon monoxide and that if these substances are reduced in amount, then so is the risk of illness". (65) The reaction to this article and its claims has drawn unusually intense scientific criticism. Indeed, the journal which published the article received numerous letters challenging the scientific validity of its conclusions. (66)

The difficulties in evaluating data on the low-yield cigarette do not stop at differences of opinion regarding the levels of one, two or even more constituents. As another researcher in the area observed when he discussed this design question, changes in constituent levels present "a tricky problem of cigarette engineering" as "there is virtually no major manipulation that does not cause a major modification in one or another perceptible aspect of smoking." (67)

Among such modifications is the possibility expressed by some researchers that smokers who switch from high "tar" to low "tar" cigarettes might modify or change their smoking behavior.

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This "compensatory smoking" hypothesis has been disputed by other researchers. The theory's adherents, however, contend that smokers tend to increase the amount they smoke by inhaling more deeply when they switch to low-yield cigarettes, by smoking further down the "rod" of the cigarettes, or by shortening the interval between puffs. (68) These researchers also suggest that individuals who smoke low-yield cigarettes may simply smoke more cigarettes. (69)

One British researcher recently summarized the "compensatory smoking" hypothesis as follows:

> In general, a reduction in the tar yield of a cigarette is associated with a fall in its nicotine yield. Not surprisingly, therefore, when smokers switch from high to low yield cigarettes they often compensate by smoking more. They may simply smoke more cigarettes a day; but -- unlike laboratory analytical smoking machines -- smokers may also vary the frequency and volume of puffing and so increase the yield from each cigarette. Alterations in the depth of inhalation may also influence the uptake of constituents of cigarette smoke. Nevertheless, the average smoker's compensation for reduced nicotine yields seems incomplete. Indices of exposure to tar and gaseous components of smoke seem to be lower when smokers switch from high to low yield cigarettes. (70)

An American researcher similarly admitted that "compensation" is "embarrassingly imperfect and incomplete to those of us trying to establish nicotine as the reinforcer in smoking" but suggested that "it may nevertheless mitigate the beneficial effects of smoking weaker cigarettes." (71) Another American researcher concluded that the "use [of low yield cigarettes] is not associated with substantially reduced smoke

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exposure . . . [because of] modification of puffing behavior, depth of inhalation, [and] blocking of smoke-dilution holes of ventilated filters." (72) A study sponsored by the U.S. National Research Council likewise suggested that changes in smoking behavior "offset to some degree the expected reductions in nicotine and tar . . . [although] smokers still may not absorb from the lower-yield brands all of the [tar and nicotine] previously absorbed from higher-yield brands." (73)

These studies have prompted public health officials in some countries to criticize the claim of maximum constituent level proponents that low-yield cigarettes are "safer" to the smoker. The U.S. Surgeon General, for example, has stated that "[c]ompensatory behavior may negate any advantage of the lower yield product or even increase the health risk." (74) Likewise, a Finnish health official concluded that "low-tar cigarettes give a much higher yield of harmful components than has been presumed" because low-yield cigarettes are smoked more "intensively and efficiently" than high-"tar" cigarettes and are "more deeply inhaled." (75) The World Health Organization recently pursued this same theme in a pamphlet for use in certain Middle-East nations. This pamphlet advises smokers that low-yield cigarettes are "no less dangerous than other cigarettes" because "the smoker tends to compensate the loss in tar and nicotine by smoking more, [and] inhaling more deeply." (76)

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On this question as on so many others pertaining to the low-yield cigarettes, however, the data are inconclusive and frequently contradictory. For example, while some researchers assert that switchers modify their smoking behavior to maintain a certain level of nicotine (77), others have suggested the possibility that smokers may actually modify their smoking at least in part to increase their "tar" intake. (78) After analyzing the latter report, the 1984 U.S. Surgeon General's Report commented, "It may be that nonpharmacologic, sensory stimulation by factors such as the flavor of the cigarette smoke may be more important than nicotine in determining smoking behavior." (79) This comment is supported by another study published by the same researchers (discussed in the 1984 Report) following their long-term observation of such switchers. They reported that smokers who switched to cigarettes with lower levels of nicotine did <u>not</u> change their smoking patterns in such a way as to maintain the same level of nicotine they had absorbed before they switched. Thus, they suggested that any change in the smoking behavior "was not determined by a need to maintain nicotine intake." (80) Two American researchers who have reviewed such research note that the data are "far from conclusive, and further research is definitely needed." They assert that "only sound methodological studies will lead to more definitive conclusions" regarding this hypothesis. (81)

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Even before such scientific and technical uncertainties were expressed, questions had been raised regarding the necessity for establishing maximum constituent levels. A 1977 study, for example, suggested that the rationale for limiting "tar" and nicotine levels was lacking:

> "Much money and effort are being spent on attempts to reduce tobacco smoking . . . . Some recent suggestions and developments include . . . cigarettes with low nicotine and tar yields, or cigarettes with high nicotine and low tar yields. The rationale for such measures is lacking since the role of nicotine and the mechanisms of pharmacologic reinforcement in tobacco smoking remain virtually unexplored." (82)

Similarly, a Belgium scientist looking back over the long-standing controversy surrounding the issue of smoking and health observed:

> "Many scientists now claim that the reduction of the nicotine content in tobacco was not necessary at all and that all the laws and regulations which still attribute the principal risks of tobacco to nicotine have become virtually obsolete." (83)

Such comments clearly demonstrate that there is no reliable scientific basis for imposing maximum tobacco smoke constituent levels. The claims of MCL proponents that low-yield cigarettes are preferable, from a public health standpoint, to other cigarettes are no more well founded than the claims that particular smoke constituents have been determined to cause the alleged health risks of smoking. The imposition of maximum constituent levels gives

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governmental sanction to the "benefits" of low-yield cigarettes posited by the MCL advocates; the relevant governments would, if the proposal is adopted, mislead consumers by suggesting that a reliable scientific basis exists for switching to low-yield cigarettes. No such basis exists.

III. CONCLUSION

There is no convincing evidence that the imposition of government-mandated maximum constituent levels will result in any claimed health "benefits". Numerous claims have been made about possible relationships between cigarette smoke constituents and the health of the smoker. However, such claims are just that -- claims which are not established scientifically. "Tar" is a laboratory product and not an actual constituent of tobacco smoke. Proponents of maximum constituent levels rely upon flawed animal experimentation in arguing that "tar" levels have relevance to the human situation. No definitive conclusions about "tar" and human disease can be drawn from skin painting studies -- these experiments involve applying "the wrong material, in the wrong form, in the wrong concentration, to the wrong tissue of the wrong animal." (84)

Likewise, no relationship between the nicotine level of a cigarette or the number of cigarettes smoked and the smoker's actual nicotine exposure has been established because of individual

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variations in puff rates, depth of inhalation, and body metabolism. Thus, science has not been able to determine with any precision how much nicotine a smoker is exposed to. Epidemiological studies can never demonstrate a causal role for nicotine in heart disease. Laboratory studies in this area are of questionable validity. As one researcher observed in regard to the conclusion of the 1981 Surgeon General's Report that changes in the composition of cigarettes over the last 15 years have not been related to declines in cardiovascular disease, this result is "clearly consistent with the proposition that amounts of tar, nicotine, and carbon monoxide in cigarette smoke have no demonstrable effects on CHD rates, and that these components of tobacco smoke are not proven etiologic (causal) agents." (85) Carbon monoxide, another constituent of cigarette smoke, is also accused by anti-smoking advocates of playing a role in the development of cardiovascular disease. The evidence for this is tentative, however, and has justly been described as "open to varying interpretations." (86)

Thus, there is no convincing evidence that the imposition of government-mandated maximum constituent levels will result in any health "benefits." Proposals to mandate maximum constituent levels lack a reliable scientific basis. For this reason, programs and proposals to limit constituent levels such as one sponsored by the Finnish Government are misleading to the smoker because they suggest that a reduction in the levels of certain constituents would

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decrease any claimed harmful effects of cigarette smoke. This possibility was noted by Dr. Matti Rimpela, a well-known antismoker, in a 1987 report on changes in Finnish adolescent health habits:

> It is possible that the increased selection of "light" cigarettes has influenced older adolescents' smoking behavior. When the dangers of smoking are equated with tar content, a decrease in tar content can lead to the belief that cigarettes are less dangerous. (87)

Such misleading and misguided legislative efforts should be avoided.

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